

* DIABETIC FOOT :

* **Definition:** It is a serious foot infection ± gangrene in diabetic pt.

* **Incidence:** 20% of D pt will have D foot & it is the most common cause of LL amputation. [20% of D ulcers ends by amput.].

* predisposing factors:

① **Vascular:** - Macroangiopathy → by atherosclerosis mainly leg vessels.
- Microangiopathy → by arteritis

NB: atherosclerosis differs from non diabetics in that
1- appears early & affect vessels below knee.
2- more progressive as it is continuous lesion (not patchy)
3- abnormality mainly in basement membr. of endothelium

② **Neuropathy:** - DM affects both sensory & motor systems:-

⚠ **Sensory:** - Sensory loss takes "Glove & Stocking distribution"
[It is due to myelin deficient synthesis] → susceptible to trauma (unware) → ulceration & infection

NB: Sensory loss is for deep sensation (vibration & joint position) before superf (pain, touch & temp.)

⚠ **Autonomic:** - (auto-sympathectomy):

⇒ Loss of ms tone → vein dilation → congestion & oedem → skin stretching → skin breakdown (ulcer) → infection.

⇒ Anhidrosis: Less sweating → dry skin → ↑ infection.

⚠ **Motor:** - mainly small ms. of foot → imbalance → deformity → ↑ trauma.

③ **Immunodeficiency:** - it is represented by:

⇒ ↓ glycosylated Ab formation.

⇒ ↓ phagocytic activity of Leucocytes.

⇒ ↓ delivery of Ab & phagocytes to site of infection (microangiopathy)

* Investigation:-

- CBE (Hb, TLC) - FBS, HgA_{1c} (glycosylated Hb), urine for sugar
- X-ray → gas under skin, bone change (osteomyelitis)
- Culture & sensitivity (from discharge or blood).
- Vascular assess (Doppler, arteriogram, ...)

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* Clinical picture:

- H/o minor trauma, with foot infection, offensive odour & gangrene or H/o complication as osteomyelitis.
- examine locally (see ulcer exam. ¹⁰⁰) and generally (DM is multi-systemic).

* Management:

① prevention & education:- control DM, good hygien, avoid trauma.

② Medical ttt:- it includes:

a- control of DM:- as infection ↑ level of glucose & vice versa ie makes difficult control so change into insulin if pt on oral hypoglycemic.

b- control of infection:- done by:-

- Debridement of necrotic tissue ^{mechanical or chemical (H₂O₂)} if after it is bleeding & oozing (ie ϕ neuropathy mainly) if no oozing & bleeding (ie vascular-ischemia) → "angiography"
- Dressing (always moist). & pus drain.
- systemic antibiotics (broad spectrum) see c/s.
- NB in chr. osteomyelitis debridement is mandatory

③ Surgery:- amputation for gangrene

④ Correct underlying diseases as anemia (to ↑ tissue O₂) and cardiac dis., Tri B complex for sensory loss.

* Stages for D.F infection:

① stage of cellulitis: skin erythema by streptococcal infection or staph (in staph is bullae formation). No Pus. No discharge

② stage of Deep skin & soft tissue infection: pt has severe pain
NB:- the D/D is compartment syndrome or gas gangrene

③ stage of acute osteomyelitis: pt has pain in bone + foul discharge

④ stage of chronic osteomyelitis: pt has continuous discharging sinus with s/s of chr. osteomyelitis

⑤ stage of fetid foot: severe inflam. of soft tissue & bone. pt has a foul smelling.

* Stages of D.F ulcer :-

- Stage 0 - preulcerative or post ulcer $\bar{e}$ fully epithelized.
 - A $\rightarrow$ pre-or post-ulcer $\bar{e}$ out cellulitis.
 - B $\rightarrow$ " " " " $\bar{e}$ cellulitis.
 - C $\rightarrow$ as A $\bar{e}$ signs of regional ischemia.
 - D $\rightarrow$ " " " " " severe ischemia.
- Stage I - Ulceration (partial or full thickness) $\bar{e}$ out deep structure
 - A $\rightarrow$ deep ulcer $\bar{e}$ out cellulitis.
 - B $\rightarrow$ " " $\bar{e}$ cellulitis.
 - C $\rightarrow$ " " $\bar{e}$ regional ischemia.
 - D $\rightarrow$ " " $\bar{e}$ severe ischaemia.
- Stage II - Ulceration $\bar{e}$ soft tissue infection $\bar{e}$ out joint involve
 - A $\rightarrow$ $\bar{e}$ out cellulitis. C $\rightarrow$ regional ischemia
 - B $\rightarrow$ $\bar{e}$ cellulitis D $\rightarrow$ sever ischemia.
- Stage III - ulceration $\bar{e}$ joint & bone involvement (osteomyelitis)
 - A $\rightarrow$ $\bar{e}$ out cellulitis C $\rightarrow$ regional ischemia.
 - B $\rightarrow$ $\bar{e}$ cellulitis D $\rightarrow$ Sever ischemia.
- Stage IV - fore foot gangrene (toes).
- Stage V - Whole foot gangrene.

• Fontain classification: for chronic ischaemia :

- Stage I $\rightarrow$ asymptomatic
- Stage II $\rightarrow$ intermittent claudication
- Stage III $\rightarrow$ rest pain
- Stage IV $\rightarrow$ gangrene

• Boyd's classif. : for degree of ischemia (claudication):

- Stage I $\rightarrow$ $\uparrow\uparrow\uparrow\uparrow$. Pain (claudi) starts on walking & $\downarrow$ by continue walking (good collateral) $\rightarrow$ mild ischemia
- Stage II $\rightarrow$ $\uparrow\uparrow\uparrow$. Pain starts on walking & gradually $\uparrow$ by walking ie $\rightarrow$ moderate ischemia.
- Stage III $\rightarrow$ $\uparrow$ - Pain starts on walking but fore the patient to stop $\rightarrow$ Sever ischemia

LYMPHATIC DISORDER

LYMPHOEDEMA

* Definition :- hypertrophy of skin & s.c tissue by chronic lymphatic obstruction.

* Causes & types :

① Congenital (1st) - [rare] manifested either $\left\{ \begin{array}{l} \text{at birth} \rightarrow \text{Milroy's dis.} \\ \text{at puberty} \rightarrow \text{Precox.} \\ \text{at adult} \rightarrow \text{Tarda.} \end{array} \right.$

② Acquired (2nd) - [common] -

- Traumatic : e.g Burns, LN block dissection.
- inflammatory : e.g Filariasis, TB lymphangitis, ..
- Neoplastic : e.g Metastasis, Lymphoma.

* Stages of lymphoedema :-

- Stage I :- soft pitting oedema.
- Stage II :- Lymphorrhoea due to rupture of lymph vessels.
- Stage III :- Non pitting oedema due to Fibrosis of skin & s/c tissue
- Stage IV :- Warty-pseudo-papillomatous formation :- ie elephantiasis.

* Complication :-

- Recurrent cellulitis & Lymphangitis (commonst).
- Lymphoedema ulcer & infected blebs.
- heavy & huge limb.
- Lymphangioma sarcoma (v. rare)

* Treatment :-

- ① Conservative ttt : [in early cases] $\rightarrow$ Rest & foot elevation, Massage, elastic stocking, Diuretics, Antibiotics & Anti Filarial.
- ② Surgical ttt :- [in chronic cases] $\rightarrow$ Removing the thick s.c tissue for cosmetic & functional reasons e.g Sistrunk operation, • Charles's (flaying) operation OR Thompson's (swiss roll) operations.

LYMPHADENOPATHY :

* Causes of Lymphadenopathy [LN enlargement] :

- ① Inflammatory :-
- ① Acute - non specific : - septic lymphadenitis .
- infection mononucleosis .
 - ② Chronic - non specific : - ch. tonsillitis & pharyngitis
- scalp infection as pediculosis capitis .
 - = specific : - Bacterial → TB. § .
- Parasitic → Filariasis
- Viral → Cat scratch, AIDS
- Protozoal → Toxoplasma.

- ② Lymphoma :-
- ① Hodgkin lymphoma.
 - ② Non-Hodgkins lymphoma.
 - ③ Burkitt's lymphoma.

- ③ Blood diseases :-
- ① Acute leukaemia
 - ② Chronic myeloid Leukemia.
 - ③ Chronic lymphatic leukemia.

- ④ others → as metastasis, collagen disease & Lipoidosis.

LYMPHOMAS

* Definition :- Primary malignant neoplasm of RES, it can be

- ↳ Hodgkin's L.
- ↳ Non Hodgkin's L.
- ↳ Burkitt's L.

I - BURKITT'S LYMPHOMA

* Definition :- The rarest type of Lymphosarcoma, it is of unknown etiology but related to EBV "Ebstein-Barr virus".

* C/P :- (Common in < 12 yrs in Western Africa), usually painless, progressive Jaw swelling is the usual presentation [may affect kidney, bone, ovary, CNS]

* Pathology :- Starry skin appearance = pale retinaculum & dark lymphocytes.

* Treatment :- chemotherapy.

	II HODGKIN'S LYMPHOMA	III NON-HODGKIN'S
incidence	- Commonst type of lymphoma.	Rare type, may associated = SLE, Sjogren's syndrome
site:	- <u>Nodal</u> ^(common) : Mainly cervical then generalized - <u>Extranodal</u> (rare): - in late stage → Liver, spleen	- <u>Nodal</u> (less common) → cervical. - <u>Extra N.</u> (More common) → abdominal (Liver, spleen, GIT mucosa,)
Pathology	- <u>N/E</u> : LN replaced by pink neoplasm w doesn't infiltrate surrounding (v. late) - <u>Micro</u> : Pleomorphism of cells + Reed-Sternberg cells : → w is pathognomic giant cell = multinuclei 2-8 in mirror image 	- <u>N/E</u> : LN replaced by white N. that infiltrate surrounding - <u>Micro</u> : - normal architecture of LN. are lost by tumour cells, lymphocyte, Retikocyte...
classific.	(Based on histological type) • Lymphocyte dominant type → Best prognosis. • Lymphocyte depleted type → worst " • Nodular sclerosing type. • Mixed cellularity type.	(Based on cell origin) • B-cell lymphoma • T-cell lymphoma • Lymphoblastic lymphoma • Histiocytic lymphoma.
C/P	• young , male > Female • Rubbery Painless LN, discrete & different ^{size} • <u>Extranodal</u> → <u>systemic symptoms</u> : High sweating, wt loss, pruritis anemia, Pel-Ebstein fever → (2 wk fever alternate = 2 wk free), alcohol → ↑ pain • S/S of pressure of LN at surrounding.	• Elderly , M > F • Firm Painless LN. (may soft → if degenerative) (may hard → if calcified) • Extranodal features in case of infiltration as Hodgkin's
t/t	- Stage I (A & B), II A → Radiotherapy alone - Stage II B → Chemotherapy (MOPP) as 6 cycles supplemented by Radiotherapy - May surgery as → splenectomy (hypersplenism) → Decompression (pressure s/s)	- Radio & Chemotherapy according to stage. - May surgery to resect LN in gastric & intestinal lymphoma
prognosis	relatively good prognosis (5 yr survival 80% if proper t/t)	very bad.

* Staging of lymphoma (Hodgkin & non-H):

- Stage I: - one group of LNs only.
- Stage II: - More than one gp. LN same side of diaphragm.
- Stage III: - LN. involved above & below diaphragm.
- Stage IV: - Extranodal spread as liver, spleen, BM, ...

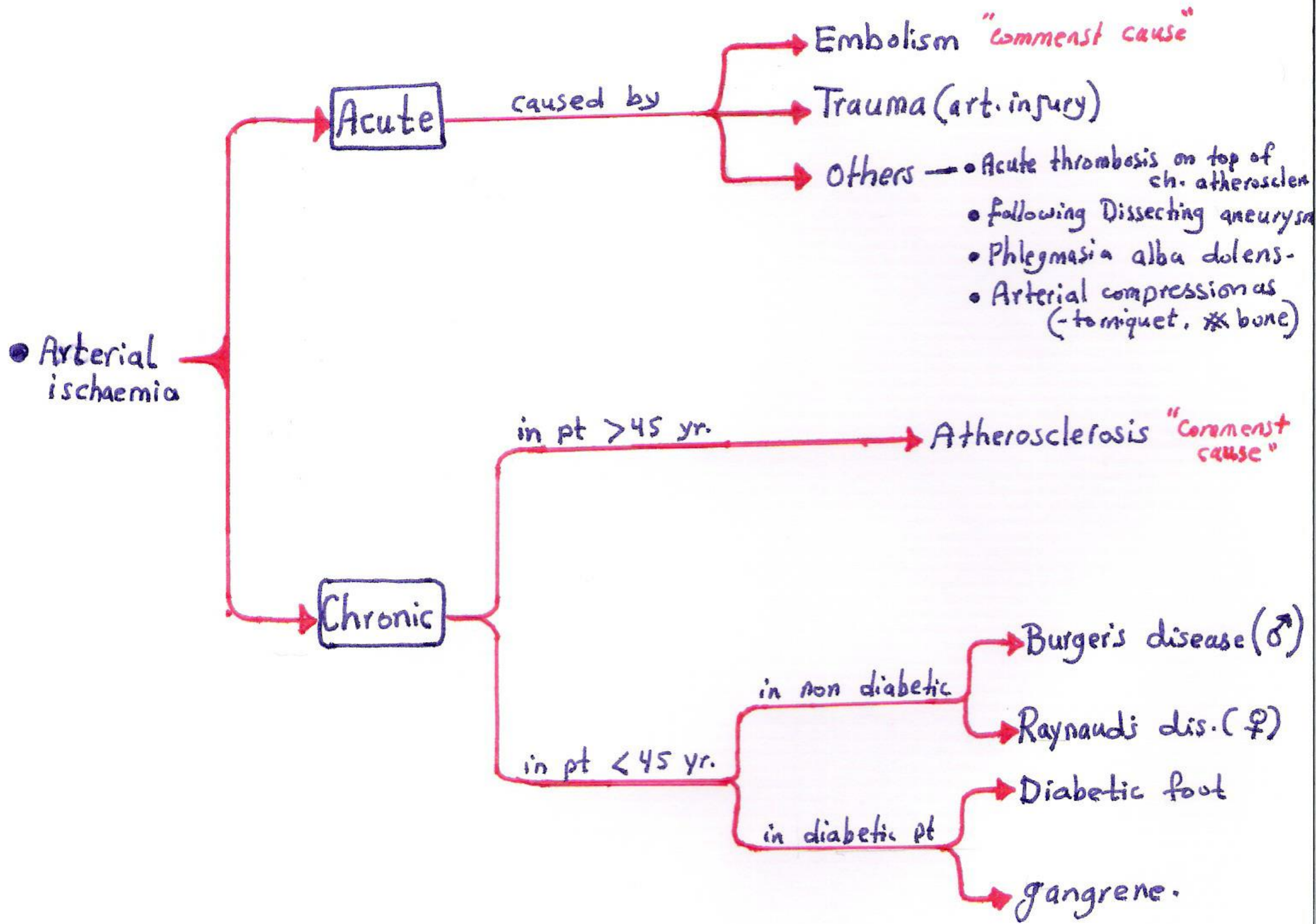
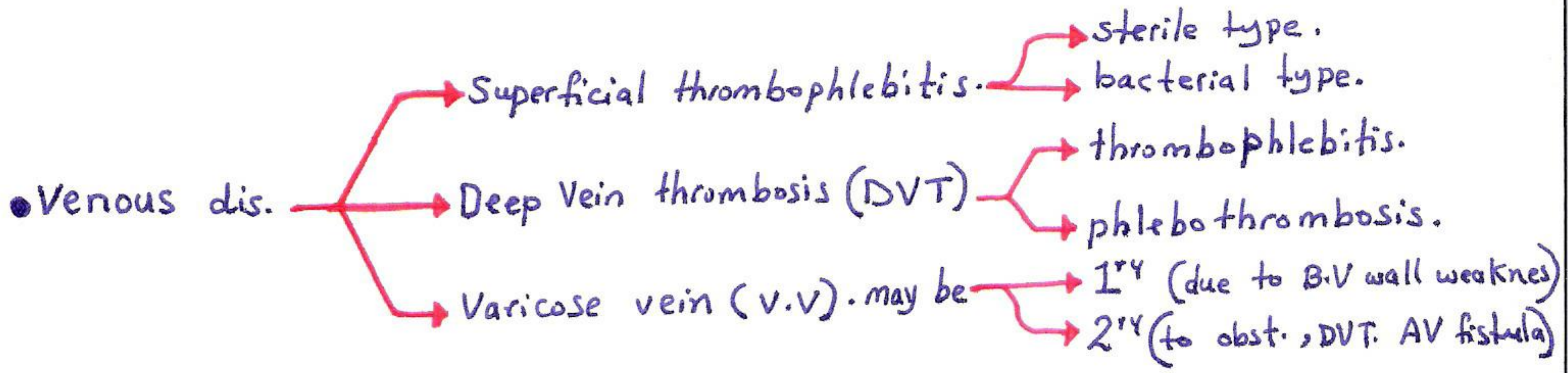
any stage may be

(A) → No systemic s/s

(B) → = systemic symptoms (one or more)

VASCULAR DISORDERS

The vascular disorders are mainly venous and arterial disorders (mainly arterial ischemia).



VENOUS DISORDERS

I - SUPERF. THROMOPHLEBITIS:

*Definition:- thrombosis of inflamed superficial veins.

*Aetiology:- ① systemic cause :- Polycythaemia, SLE, ulcerative colitis, Mondor's disease
② Local cause :- Varicose vein (v.v), Burger's dis., trauma iatrogenic (i.v infusion)

*Types:- ① Sterile type ② Bacterial type

*C/P :- -Symptoms: pain + FHMA (Fever, Headache, Malaise & Anorexia).
-Signs:- Tender Cord like structure ± overlying skin redness.

*Treatment:- ① Plastic bandage & underlying cause ② Analgesia & A.B (if bacterial type).

N.B.: thrombo-phlebitis Migrants [Trousseau sign] :- associated with internal carcinoma (as stomach & pancreas) due to increased viscosity of blood.

II - DEEP VEIN THROMBOSIS

*Definition:- thrombosis of deep veins, occurs more in calf veins

↳ valveless & sinusoids.

*Aetiology:- [Virchow's triad]:

① Vessel wall changes (Endothelial damage): due to trauma or inflammation.

② Blood flow changes (Venous stasis): in heart failure or compression as in pregnancy & tumour.

③ Blood composition changes (Hyper coagulability): due to:

- Oral contraceptive pills or polycythemia.
- deficient of - Antithrombin III or natural anti coagulant (as protein C & S).

*Risk factors → Other causes as old age & previous H/O DVT.
→ Commonly (>50%) after ^{major} operations [as *NOF, prostatectomy].
→ obesity & female on contraceptive pills.

*Types of DVT :-

The DVT may be due to Phlebotrombosis or thrombophlebitis.

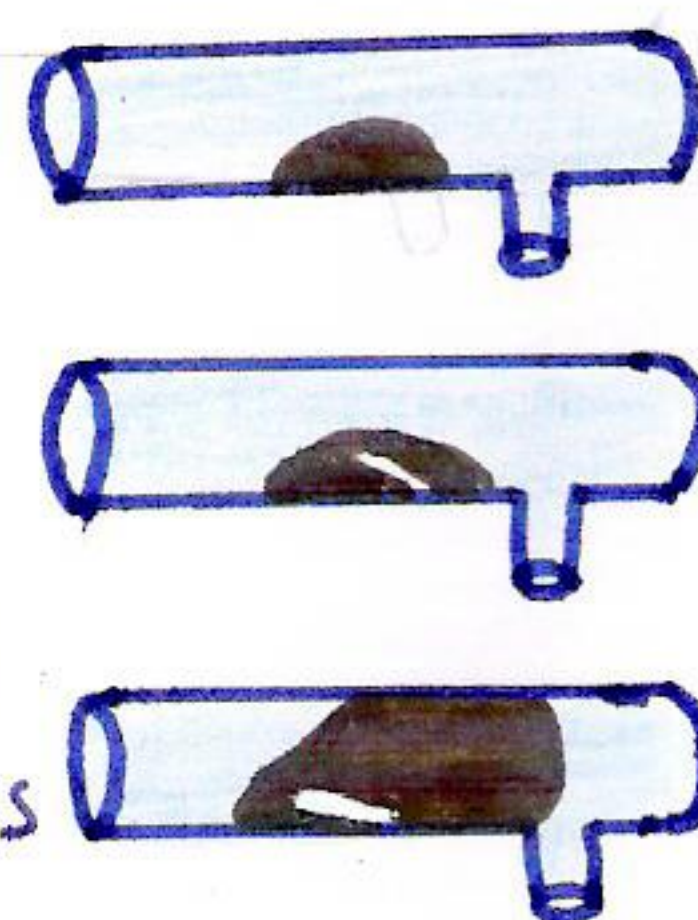
	Phlebo-thrombosis	thrombophlebitis
Definition	thrombosed <u>un</u> inflamed veins	thrombosed inflamed vein
Causes	Stasis or hyperviscosity	Draining inflamed organs
Site	commonly leg veins	commonly pelvic veins
Size of 1 st thrombus	Small	Large
Emboli	Common & Sterile	Rare & infective

*Pathogenesis:-

= Early stage: ①- Platelet adhere to endothelium of B.V forming "Gray cluster", mainly in calf veins.

②- fibrin & RBCs deposited in a layer between platelets forming "Line of Zahn".

③- After total occlusion of vein, thrombus spreads & the "Jelly-Like propagated thrombus" spreads as far as the near major tributary.



NB: At early stage pulmonary embolism is common because of loosely attached thrombus.

= Late stage: ④- the thrombus becomes tightly adherent → destruction of valves & occlusion of lumen = Post-phlebitic Limb. (see next)

⑤- by fibrinolysis → "Recanalization", (but valves destroyed permanently)

*Clinical picture [C/P]:

- The classic picture is ①-Pain :- aching discomfort commonly at calf. (25% are symptomatic)

②-Swelling:- ie oedema [reliable sign].

③-Tenderness:- on pressing muscle against bone.

= pt may be asymptomatic either silent or unexplained fever & ↑HR.

= pt may presents by pulmonary embolism as a 1st manifestation

= N.B - Symptoms & signs depends on the level of thrombosis

- Homan's sign (not used nowadays bec. may → embolisation) which is a +ve calf pain by sudden dorsiflexion.

NB:- the commonest vein for DVT is calf vein & rarest is IVC.

- The iliofemoral thrombosis may result in:

	① Phlegmasia alba dolens	② phlegmasia cerulea dolens
Definition	Painful white limb (Phlegma = inflammation)	- Painful blue limb.
Cause	Partial venous obst. (Less severe)	- Complete venous obst. (more severe)
C/P	Pallor: due to associated artery spasm	- Cyanosis (no venous return)
Complicat.	Coldness & ↓ pulsation	- venous moist gangrene.

* Investigation for DVT:

- ① Doppler U/S:- in completely obstructed leg vein shows absent flow & incompetent perforators "accuracy 85%"
- ② Coloured Duplex: shows an image of veins & bd flow direction "accuracy" 90-100%
- ③ Venography:- thrombus appears as a filling defect. [less used now].

* Complication of DVT:

- ① Early:
- ① Pulmonary embolism.
 - ② Venous gangrene (in phleg. cerulea dolens).

- ② Late:-
- ① Secondary V.V (varicose vein).

② Chronic venous insufficiency "post-phlebitis limb or syndrome"

- It is due to iliofemoral thrombosis with destroyed valves
 this → reflex of blood (through incompetent perforators) → high pressure in superficial veins especially during walking "Walking venous hypertension".

- In this case it • Non pitting oedema (fibrosis)

• Venous ulcer w may → Malignancy "Morgagni ulcer"
 → Periostitis
 → Talipes Equinov.

Blow-out syndrome

* Treatment of DVT:

• Prophylactic: General measures as adequate hydration, avoiding calf pressure & early post-op. mobilization.

- stop oral contraceptive pills (some school stop it 6 wk pre-op)
- othe school recommend heparin prophylaxis
- slight leg elevation (15-20°) after operation.

= prophylaxis for high risk patient include:

- Low dose heparin (mini-heparin) :- 5000 IU (s/c) : used 2 hr before operation & 12 hourly for 7 days or until mobilization.
- Low Molecular weight (LMW) heparin :- once daily.
 - it has lower risk of bleeding so more popular
- Calf compression devices intraoperative.
- graded compression stockings before operation until mobilization.

• **therapeutic ttt** : (anticoagulant).

- Giving heparin (i.v or s.c) or warfarin (oral), usually start by heparin then continue warfarin after.

	Heparin (i.v or s.c)	Warfarin
- Action	↑ activity of fibrinolysis	- Block synthesis of prothrombin & Factors VII, IX, X
- Dose monitoring	- APTT (must kept at 2.5 times the normal)	- PT (prothrombin time)
- Antidote	- Protamine sulphate	- Vit K.
- Complication	- Bleeding (over dose) - Failure of response (heparin resistance)	- Bleeding, peptic ulcer, - interaction with aspirin.
- Dose:	① i.v 5000 IU / 4 hourly. OR ② i.v 5000 IU plus then infusion of 5% glucose @ rate at 20-30 IU/Kg/hour. ③ s.c calciparin 150 IU/Kg/12 hr.	- Oral 10 mg initially then 5 mg for 5 days

NB :- Fibrinolytics (as streptokinase, urokinase & TPA) not used widely because of severe bleeding, allergy & very expensive

= Surgical Rx may be needed as venous thrombectomy in case of phlegmasia cerulea dolens by Fogarty catheter.

III - VARICOSE VEIN :

*Definition:- Dilated tortuous, elongated veins (superf. v. of lower limb).

*Anatomy:- The venous system of lower limb is either:

- ① superficial system → (Great & lesser saphenous veins)
- ② Deep system → (venae comitantes to arteries & venous sinuses inside calf ms "soleus").
- ③ Connecting system → connect superf. to deep.
 - Indirect communicators; ie from superf. → ms → deep veins
 - Direct comm. (perforators)
 - 1) three ankle perforators 2, 4 & 6 inches above medial malleolus (5, 10 & 15 cm)
 - 2) One perforator just below knee.
 - 3) one " at mid thigh.
 - 4) Sapheno-femoral junction
 - 5) Sapheno-popliteal → (in lesser saphenous)

*Types & causes of V.V :

	1 st V.V (non-obst.).	2 nd V.V (obstructive).
Causes :	- Congenital weakness of the bl. vessel wall → Dilated - vein → incompetent valves - Precipitated by long standing & sitting.	- obstruction of vein flow: e.g pregnancy, fibroid, ovarian cyst or any pelvic mass - Valve destruction as in DVT. - high flow & pressure as A.V fistula
C/P	- minimal oedema & skin changes & usually Bilateral, heaviness pain - The vein usually tubular  or sacular 	- Marked oedema & skin changes & usually Unilateral, bursting pain - The vein usually spider  or serpentine 
Past history	- No H/O DVT. - F/H of weak mesenchyme as • Varicocele • Piles • Visceroptosis • Hernia • Kyphosis • Flat foot	- H/O underlying cause as DVT

* Investigation of V.V. :-

- ① Venography :- shows patency & size of vein, valves & Blow out.
- ② Doppler u/s & Duplex → most imp.
- ③ Look for cause → abd. & pelvic u/s, Arteriography (A-V fistula)

* Complication of V.V. :-

- (A) Venous compl. →
 - 1) Hemorrhage.
 - 2) Superf. thrombophlebitis.
 - 3) Phlebolith (calcified thrombosed vein).
- (B) Skin compl. →
 - 1) Brown discoloration → due to extravasated hemosiderins
 - 2) Dermatitis → due to irritant hemosiderin.
 - 3) Eczema → due to scratching dermatitis.
 - 4) Oedema → pitting, but later non pitting.
 - 5) Ulcer → Common after post-phlebotic cases.
 - 6) Malignancy → at ulcer site "Marjolin's ulcer."

* Treatment of V.V. :- "1st & cause if present"

① Conservative ttt :

- Indication :- early 1st V.V, pregnant, pt waiting, unfit or refusing surgery
- Methods :- below knee stocking, elevation of leg, regular exercise & avoid long standing & sitting.

② Injection-compression sclerotherapy :

- Aims to occlude the vein by fibrosis
- Indication :- 2nd V.V (spider type) & residual veins after operation.
- Contraindication :- 2nd V.V & DVT, pregnancy & acute ^{septic} thrombophlebitis
- Method :- inject the vein after emptying & apply firm elastic bandage for 2 wk (some schools for 6 wk).
 - Using either ① 3% Na tetradecyle sulphate.
 - ② 5% Ethanolamine oleate. or
 - ③ 5% Na Morrhuate.
- Use a small dose (1 ml) and inject only one vein & others at other visits & advice exercise.

③ operations of V.V. :-

- Indication :- 1) Saphena varix & Blow out.
2) repeated superf. thrombophlebitis.
- Subcutaneous stripping of long saphenous is the common operation, done by ligating long saphenous & its tributaries then stripping of whole long saphenous.
- NB : Ligation of vein & out stripping is called Trendelenberg's operation done in case of sapheno femoral incompetence.

NB : Saphena varix : [if 1st V.V.] : is a saccular dilatation shows expansile impulse on cough at sapheno-femoral junction.

* Examination of pt. w/ V.V. :-

- ① General Exam :- look for cause (in 2nd V.V) and look for signs of weak mesenchyme (in 1st V.V).
- ② Local Exam :- pt must be standing & exposed.
 - A Inspection :- for side (uni- or Bilateral), site (below or above knee) shape (tubular, spider, ...), skin over (as pigment, ulcer.) swelling (oedema) and skeletal changes (as Talipes equinus)
 - B Palpation :- V.V are soft compressible, but may found tender cords (thrombophlebitis), thrill (A-V fistula)
 - C Percussion :- "Schwartz percussion" : percussion of vein proximally by index finger & relieving by another index distally if the wave transmitted distally → ie incompetent valve.
 - D Auscultation :- in A-V fistula → continuous machinery murmur.
 - E Special test :-
 - To test blow out, (incompetent perforator) →
 - ① Trendelenberg test.
 - ② Multiple Tournique test.
 - ③ Manual localization test
 - ④ Fegan's test.
 - To test deep veins (patent or closed) →
 - ① Perthe's test (not done)
 - ② Modified Perthe's test.

* Special tests for V.V :

(A) To detect blow out = "incompetent perforators"

① Trendelenberg test:-

- pt lies down, empty the vein (massage) & apply tourniquet just below saphenous opening & ask pt to stand up
- Result
 - slow filling from below → Normal
 - Rapid filling → blow out "incompetent perforators"
 - No filling, so remove tourniquet → Rapid filling from above → ie sapheno-femoral incompetent.

② Multiple tourniquet test:-

- pt lies down, empty the vein, & apply ³ tourniquet one just below saphenous opening, other above knee & one below knee & ask pt to stand up
- Result
 - Rapid filling of any segment means blow out in that segment removed.



③ Manual localization test "two fingers test"

- pt stand & press by index at the great saph. vein & by other index empty the vein in opposite direction
- Result: if rapid filling between your index fingers → blow out



④ Fegan's test:-

- pt stand & mark the varicose veins & lie the pt down & detect the defect of deep fascia "blow out"

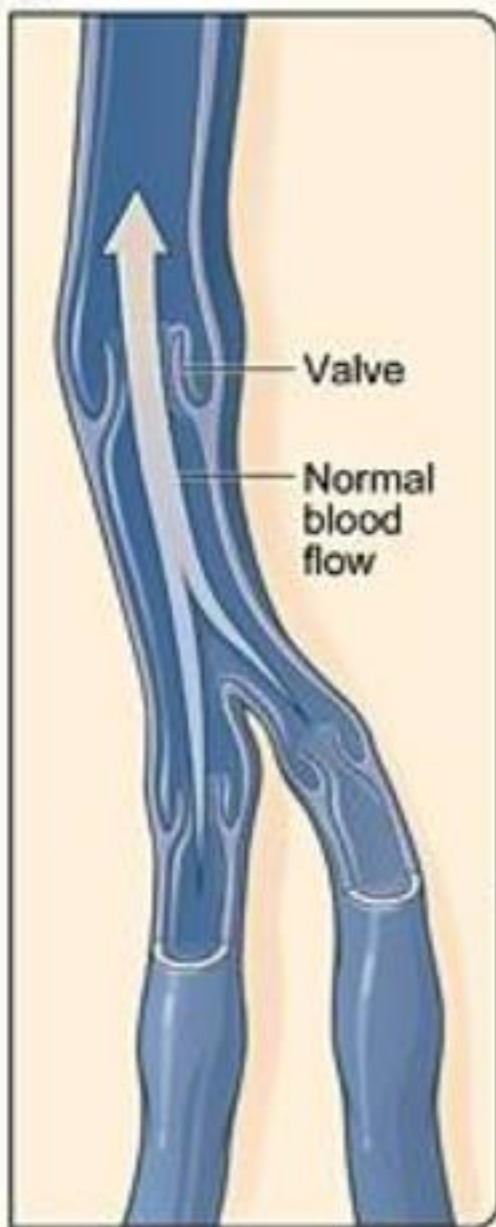
⑤ To test the deep veins if patent:-

① Perthe's test:- (not done): it depends on pain.

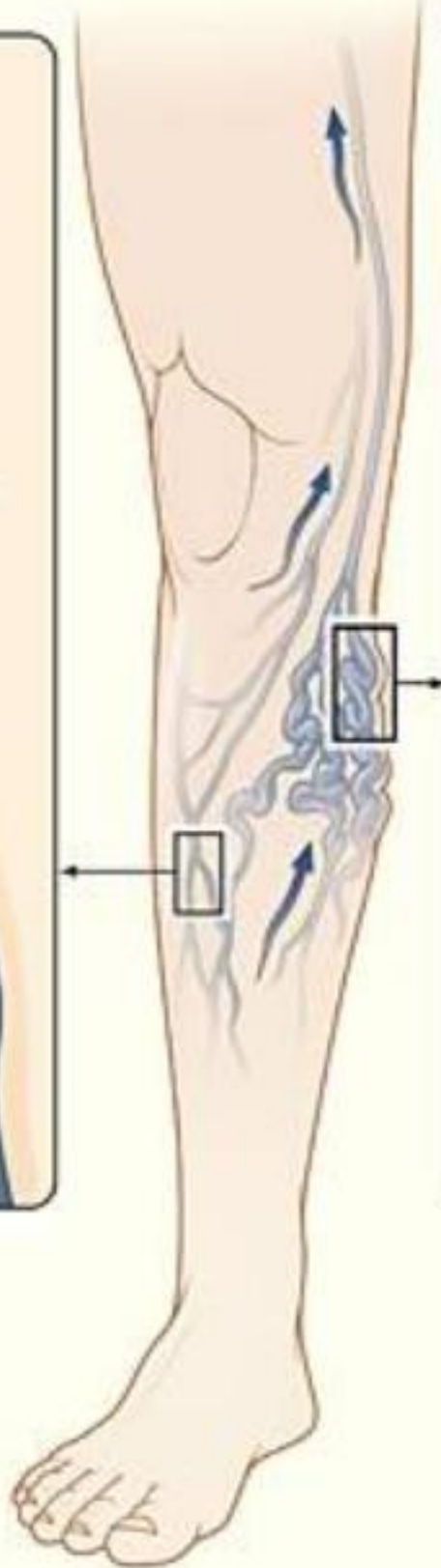
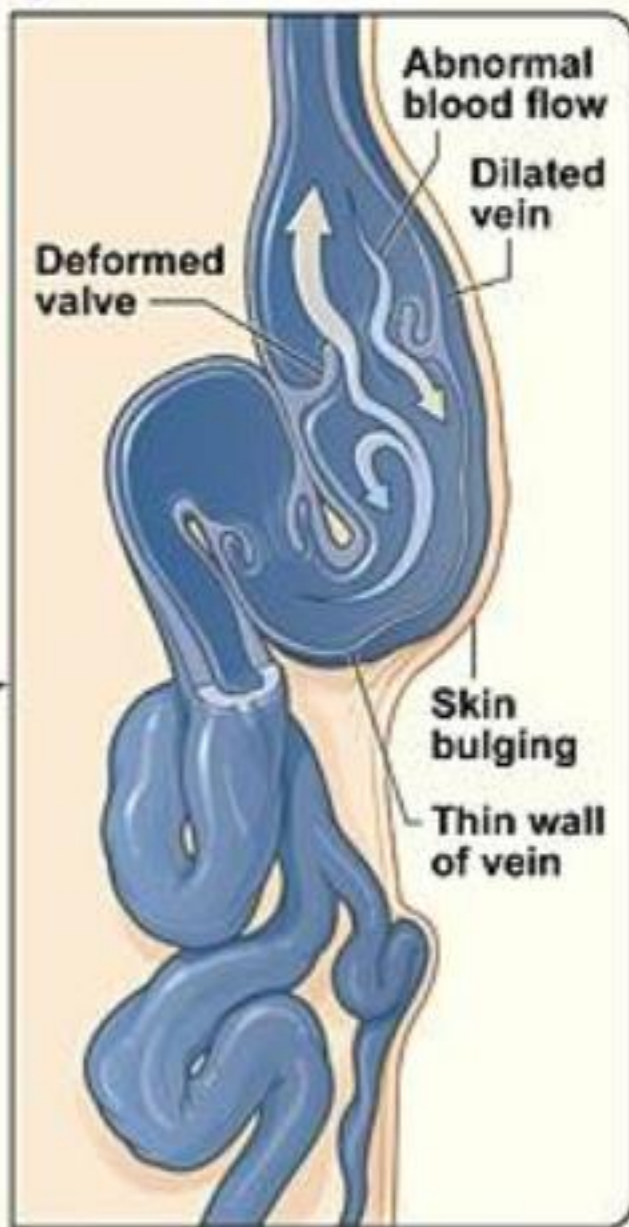
② Modified perthe's (Ochsner's test):-

- pt stand & tourniquet applied below saphenous opening & ask pt to walk insitu for 10 min
- Result:
 - if engorge (prominent) veins → occluded deep system
 - if shrunken veins → patent deep system.

A Normal vein



B Varicose vein



"Chronic Leg ulcers"

* **Definition:-** Ulcers is a discontinuity of the skin.

* **Classification:-** ① Congenital:- e.g. spherocytosis & sickle cell anemia.

② Traumatic:- Trauma & bed sores

③ Inflammatory: T.B & Φ ulcers.

④ Nervous:- Neurotrophic ulcer (e.g. Φ neuritis)

⑤ Lymphatic:- Lymphoedema ulcer.

⑥ Arterial:- ischemic ulcer.

⑦ Venous:- venous ulcer.

⑧ Neoplastic:- Marjolin ulcer.

* **C/P:- inspection:-** - Number of ulcers, site, size, shape.
- Margin, edge, floor & discharge

- **Palpation:-** - Temp., tenderness, base (soft or hard, fixed or not)

* **How to differentiate:**

• Venous ulcer $\rightarrow$ H/o varicosities, sloping or punched out edge, brown discolor usually at medial side above med. malleolus ↓ pain by foot elevation

• Arterial ulcer $\rightarrow$ H/o claudication, ↓ pain by foot dependency with deep ulcer, usually in foot.

• Neoplastic $\rightarrow$ Raised everted edge, hard & fixed base & hard LN

• inflammatory $\rightarrow$ Undermined edge  in TB & punched out in Φ 
usually at metaphysis of Tibia in TB & middle 2/4 of tibia in Φ with caseous material in TB

• Traumatic $\rightarrow$ Punched out edge, usually at middle 2/4 of tibia

• Congenital $\rightarrow$ usually at middle 2/4 of tibia.

• Neurotrophic $\rightarrow$ usually at sole of foot with callosities around

• Lymphatic $\rightarrow$ usually at dorsum of foot & associated with swollen limb & lymphorrhea.

[NB] sloping ulcer  usually indicate healing (granulation).

• Bed sores (Decubitus ulcer) usually at pressure sites as heels, sacrum, ischial tuberosity, greater trochanter & scapular blades

$\rightarrow$ the 10/5

ARTERIAL DISORDERS

- The arterial disorders may be
 - ▀ Ischaemia (acute & chronic)
 - ▀ Aneurysm.
 - ▀ Gangrene
 - ▀ Arteriovenous fistula.

ACUTE ISCHAEMIA

*** Definition:** - Ischaemia:- is a ↓ed arterial supply sufficient to interfere w nutrition & function of the limb.
- Acute ischaemia is a sudden total occlusion of artery.

NB:- Acute ischaemia is more serious than chronic because No time for development of collaterals.

- * Aetiology:**
- ① Embolism "the commonest cause".
 - ② Trauma (arterial injury) "incidence ↑ing nowadays".
 - ③ Others: e.g - Phlegmasia Alba dolens.
 - Arterial compression (bone, tourniquet, Ligature, ...)
 - Complicating dissecting aneurysm.
 - Acute thrombosis on top of atherosclerosis.

*** Pathophysiology:** - Sudden artery occlusion → Reflex spasm of nearby vein which → tissues loaded w blood → gangrene (moist aseptic)
- Muscle irreversible damage occurs within 6 hrs while that of skin within 24 hrs.

- * Clinical picture:**
- ① **Pain:** - sudden, bursting or cramp like ↑ by movem. or warmth.
 - ② **Pallor:** - 1st pale then mottled cyanosis & may → gangrene
 - ③ **Parasthesia:** - due to nerve ischaemia (1st ^{skin} hypoaesthesia then anaesthesia)
 - ④ **Paralysis:** - e.g paraplegia in aortic bifurcation occlusion.
 - ⑤ **Pulselessness:**
 - ⑥ **Progressive coldness.**

Embolitic Ischemia

Definition

- Sudden impaction of an embolus in a narrow blood vessels.

Incidence

- Commonest esp. in Cardiac pt.

Aetiology

- Embolus usually from

- Lt atrium → MS & AF
- Lt ventricle → recent MI.
- Valves → subacute bact endocarditis
- Paradoxical embolus → in ASD, VSD
- Aorta → aneurysm.
- Atheromatous plaques → atheroma
- Rarely → fat, air, clot, Tumor cells

- Site of impaction is vessel bifurcation due to

- ↓ diameter
- slowing of bd. flow
- Turbulence of flow

C/P

= 6 Ps ± signs of heart problem

Investigation

- Detect cause → ECG, Echo, ...
- Detect art. obst → Angiography.
- Doppler uls
- Duplex scanning

Treatment

- ① Urgent Embolectomy :- done under local by Fogarty balloon catheter & give preop. heparin (prevent thrombus propagation) & postoperative.
- it must be within 6 hrs;
if after called delayed embolectomy where it only lowers level of amputation
- ② Amputation :- in established gangrene
- ③ Fibrinolytics :- ??? trial (not effective)

Complication of Embolectomy : are

- ① Sudden death by pulm. embolism.
- ② Compartment syndrome → do fasciotomy.
- ③ Reperfusion injury as ↑ K⁺ & Myoglobin from dead ms → cardiac arrest (K) or renal failure (myoglobin)

Traumatic Ischemia

- Sudden interruption of artery supply of a limb by injury

- Common nowadays (RTA, war...)

- ① Open injury → stab, bullet, ...
- ② closed " → * as subcondyl * of Femur or humerus

- Complete division of artery  bleeds profusely but stops or ↓ soon as V.C with contract. of intima & media (no puls distal)

- Partial division : not stopped bec of contraction & retraction of arterial wall, Puls is weak.

- Injury without bleeding occurs in ① art. spasm as in irritation by missile around. 
② Art. contusion ± thrombus 

= 6 Ps + H/o trauma.

- If shocked pt → resusc then immediate surgical exploration
- Cold cases → X-ray (bullet, ...) angio, Doppler, Duplex.

- ① If shocked pt → resusc & ABC assessment
- ② Control bleeding by compression or surgical
- ③ Immediate exploration.
- ④ Fasciotomy (to prevent the compartment syndrome)
- ⑤ In arterial spasm & contusion excision & venous graft done
- some try spasmolytic (e.g papaverine) & heparin in spasm
- ⑥ Rx of * bone, antibiotics, anti tetanus, ...

CHRONIC ISCHAEMIA

* **Definition** :- Slowly progressive arterial obstruction.

* **Aetiology** :-

- ① Atherosclerosis "commonest cause."
- ② Arteritis : eg Burger's disease.
- ③ Vasospastic dis. eg Raynaud's disease
- ④ Diabetic foot & gangrene.

* **ATHEROSCLEROSIS** :

* **Definition** :- Degenerative arterial disease due to Aging process affecting mainly Large & medium sized arteries.

* **Incidence** :- old (>40 yr) (male > female)

- Risk factors : heavy smokers, hypercholesterolemia, hypertriglycerides, hypertension, obesity & +ve family history.

* **Pathology** :- the 1st pathology called "Atheroma" which is elevated yellow patches at intima & subintimal ↑ in Lipid & CT and at media & adventitia & fibrosis.

* **C/P** :-

- ① **Pain** :- intermittent claudication or rest pain.

- ② **Pulse changes** :- artery palpated (thick), ↓ amplitude or absent.

- ③ **Progressive coldness** :- may be False warm as ① covered ② infected ③ sympathectomy ④ DM "autosympathectomy."

- ④ **Cutaneous changes** :- hair loss : Nail → brittle ; skin → dry, scaly with ulceration, infection at pressure sites.
- paraesthesia (gradual loss), tingling & numbness.

- ⑤ **Colour changes** :- the skin colour of lower limb may be:

- Normal colour → mild ischemia
- Fixed change → severe ischemia (pregangrene) gradually
- Postural change → Do "Burger's test" → elevate limb -
 - normally no change on elevation
 - Pallor at elevation
 - cyanosis at lowering } ischemia

Burger's angle = angle at which limb becomes pale on elevation from horizon
• smaller the angle, advanced ischemia

* Investigation of chr. ischemia:

① Clinical :- to detect degree of pain [claudication time, distance & rest time]
- to detect colour change - [Burger's test & angle]
- capillary filling test
- venous filling test } see later

② Laboratory :- Urine (for sugar), blood (CBC - anemia, FBS - DM, Lipid profile)

③ Radiological & instrumental

• Arteriography (the main) - to show
- condition of artery
- site & length of obst
- collateral condition
- Distal run off (distal flow after obst.)
- Done by Seldinger's needle.

• Doppler u/s :- to show : bd flow, degree of ischemia & site of obst
- to measure "Ankle-Brachial pressure index" which is a ratio bet. pressure of ant. tibial a & brachial a normally ≥ 1
→ • if < 0.9 → mild ischemia to moderate
• if < 0.7 → Severe ischemia
• if < 0.3 → Impending gangrene

• Coloured Duplex : (most imp. invest) → imaging & blood flow measure

• ECG & stress ECG → for coronary & myocardial condition.

* Treatment of chr. ischemia.

① Conservative :- if mild ischemia or unfit pt.

- relief symptoms by improving general health (anemia, heart lesion)
- relief pain & careful washing & drying of ischemic limb
- improve bd. supply by - VD drug as Torental or PGE₁
- antiplatelet as aspirin or Persantine.
- stop smoking, regular exercise, control of DM & HbT.

② Endovascular surgery: for localized obst. ^{< 10 cm} in large or medium a but may restenosis, done either by.

[A] Percutaneous transluminal balloon angioplasty (PTA) :-
balloon catheter to dilate stenosed part

[B] Arterial stents :- after PTA to keep lumen patent

[C] LASER angioplasty :- destruction of atheromatous plaques.

③ Surgical options:- [Indicated with rest pain + Run off]

Ⓐ Thrombo-endo-arterectomy:-

- for localized lesion in large artery.
- Arteriotomy done & removing thrombus & thickened wall (intima) & after closure patent lumen will epithelize

Ⓑ Arterial by-pass:-

- for multiple lesion in large medium arteries
- e.g - Aorto-iliac & Femoro-popliteal bypass [anatomical]
- Axillofemoral & femoro-femoral bypass [extraanatomical]
- [NB]** extraanatomical done in unfit pt as in COPD, ... the graft used is natural (saphenous) in medium a or synthetic (eg Teflon or Dacron) in large a

Ⓒ Profundo plasty:- ie dilatation of profunda in case of super. femoral block.

Ⓓ Lumbar sympathectomy:- [Rest pain + No Run off] or after amputation to improve wound healing.

- done either surgical (L2.3 ganglia) in fit pt or chemical by xylocain 1% (temporary) or phenol 5% (permanent)

Ⓔ Amputation:- either

- Conservative amputation: at line of demarcation.
- Urgent high amputation: if spreading gangrene, Toxemia done by Above or below knee amputation.

* ARTERITIS:

The arteritis may be

- Burger's disease.
- infective arteritis.
- Takayasu's arteritis (autoimmune, young ♀)
- Polyarteritis nodosa (collagen dis. ass. w SLE, scleroderma)
- End arteritis obliterans (\$, rarely TB) → rare now bec. of good ttt.

* BURGER'S DISEASE:

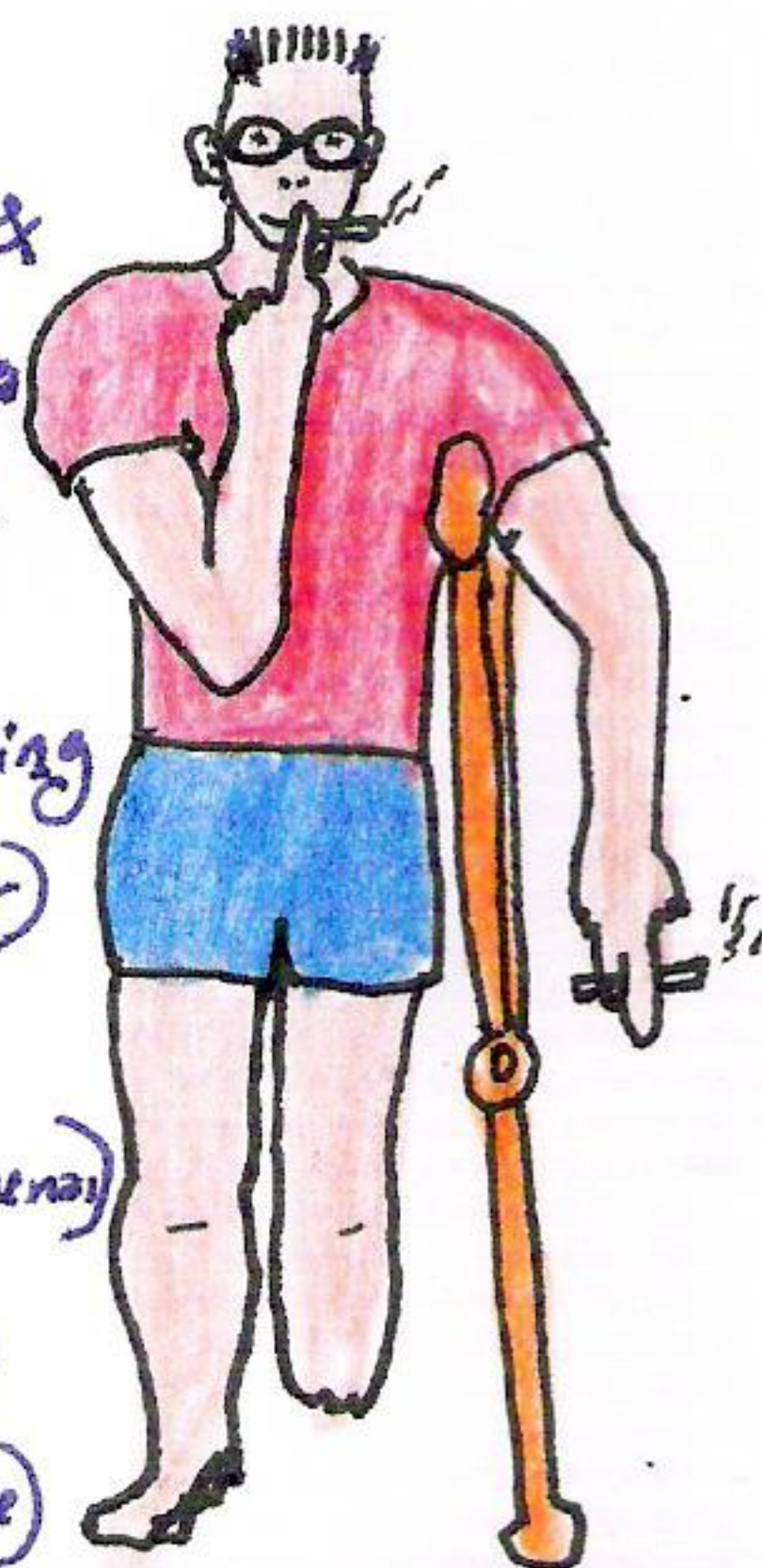
"Thrombo-Angitis Obliterans"

* Definition: Inflammation & thrombosis of small arteries & veins & perivascular fibrosis (blending vessel & nerve into one mass → early neuritis & severe rest pain).

* Aetiology: unknown, may be started by spasm & then thrombosis → fibrosis, spasm is due to smoking

* C/P → it is disease of heavy smokers, ♂, young (<40yr) involving upper & lower limbs with early rest pain & limited gangrene (+ Raynaud's phenomenon)
foot claudication, sup. thrombophlebitis

* Treatment: - stop smoking & sympathectomy → main ttt
- may conservative amputation (bypass no value)



NB: Burger's dis. differ from atherosclerosis where atherosclerosis is dis. of old age (>40yr) more in male (but burgers 95% are ♂) & no upper limb ischemia, late rest pain, claudication & the ttt. is mainly bypass.

* VASOSPASTIC DISORDERS:

RAYNAUD'S DISEASE:

* Definition: vasospastic dis. affecting digital arteries, ^{bilateral} young ♀, in cold

* Aetiology: Unknown:- but may be cold oversensitivity (not sympathetic oversensitivity as sympathectomy give early relief then recure).

* C/P: → • Pallor (due to spasm) & dull pain then
• Cyanosis (spasm → ↑ metabolites → congestion), burning pain then
• Rubor (relax. of artery & attach ends) → pain disappear.

→ pt is normal between attacks.

• Late stages → trophic changes (Hair loss, Nail brittle, dry scaly skin)
superf. ulcers & dry gangrene in finger tips.

* Management in Raynaud's dis. (success in early stages)

- Conservative treatment [avoid cold + Trental = vasospastic drug].
- Sympathectomy: in sever cases but after months mild sensitivity to cold returns.

NB: Raynaud's phenomenon :- is of known cause ie 2^{ry} to

- Systemic dis. → Collagen dis (scleroderma), myxoedema, ...
- Compression syndrome → e.g carpal tunnel, thoracic outlet syndrome
- Occupational → vibration (drill, chainsaw), piano playing, typing

* **GANGRENE** :

* Definition:- Death & putrefaction of a Macroscopic tissue in a living person

- * Aetiology:-
- Arterial obst. (acute or chr.).
 - Venous gangrene (phlegmasia cerulea dolens).
 - Neuropathic " (Leprosy).
 - Infected " (I^d gangrene, Gas gangrene).
 - Traumatic " (injury, Bed sores).
 - Physiochemical " (Burns & Frost bite).

NB:- Atherosclerotic (arterial) & infected are the most common

- * Types:-
- Dry gangrene: with chronic ischaemia
 - Shrunken, wrinkled, Hard limb, no odour
 - Moist gangren: with acute isch. (chr. isch. on top of oedema as HF)
 - Swollen, oedema, toxemia, offensive odour
 - Can be infected → very rapid spread gang
- Usually dry gangrene has line of demarcation & line of separation with band of hyperemia & anaesthesia.

* Treatment:- Amputation; either

- Conservative amputation: if good adjacent bd. supply with line of demarcation & separ. clear
- Urgent high amputation:- in spreading gangrene, toxemia either - Above knee amb. (AKA) or below knee (BKA)

* ANEURYSM:

* Definition: arterial aneurysm is a sac filled by bd. communicating with lumen of an artery.

* Classification: classified according to:-

- I- structure**: ①- True aneurysm:- wall of aneurysm is stretched wall of the artery (ie formed of 3 layers)
 ②- False aneurysm:- wall formed of fibrous tissue due to organization of pulsating hematoma.

II- shape: ① Fusiform. ②- saccular ③- Dissecting.

III- Aetiology: ①- Congenital:- e.g Berry's aneurysm (in circle of Willis)

②- Acquired:- **A**- pathological: commonest causes:

- Atherosclerotic (commonest) subacute inf end
- Mycotic aneurysm → infected emboli in SIE
- Collagen dis → Marfan's synd., Ehler's dis

B- Traumatic → weak wall → True aneurysm
 → pulsating hematoma → False "

* Clinical picture: - Usually asymptomatic, or s/s of underlying cause
 o/e:- pulsating round smooth mass (↑ by distal pressure & ↓ by proximal pressure)
 moved side to side (but not along course of the artery)

* Complication: ①- rupture → shock.

②- infection.

③- Thrombosis & Embolism.

④- Distal ischemia

⑤- Compression symptoms

Vein → oedema & varicosities.

Artery → ischemia

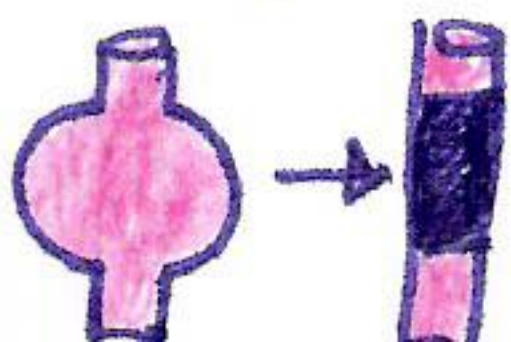
Nerve → sensory changes

Bone → erosion of bone.

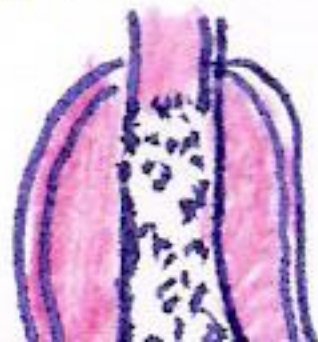
* Investigation: - Plain X-ray (calcifi-), arteriography (useless if clotted aneurysm)
 - Sonar & CT scan (the choice) & Duplex scanning.

* Treatment: - If asymptomatic & < 5cm → follow up & sonar/6 months
 - if > 5cm → surgical graft either

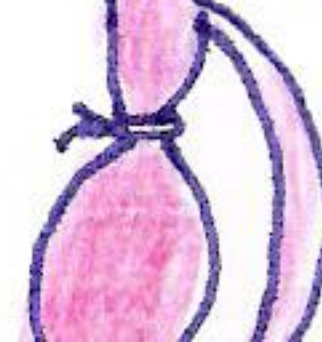
Excision + Graft
(classical)



Graft inside aneurysm
(for > 5cm)



Excision & by pass graft
(if surrounding imp. structure)



Abdominal Aortic Aneurysm: "AAA"

- The AAA is the commonest arterial aneurysm,
- It affects male > Female, age 50-60 yrs (atherosclerosis)

* Clinical picture: 75% are asymptomatic & may be discovered accidentally as pt may complain of vague abd. pain or chronic back pain [due to bone compression & interfering with vertebral blood supply] misdiagnosed as disc prolapse.
- pt may be presented by symptoms of rupture: i.e. acute (sudden) upper abd. pain at back or flanks and s/s of shock.

* invest. , treatment → see aneurysm.

* ARTERIO-VEINOUS FISTULA

* Definition: Abnormal connection between an artery & vein.

* Types & aetiology: - A-V fistula may be:

- ① Congenital: usually small & multiple, more in lower limb → Giant limb.
- ② Acquired: Trauma (stab, bullet...) or iatrogenic (hemodialysis, arteriovenous).

* Clinical picture: pain & swelling in area of fistula with enlarged tortuous artery & veins, ↑ed skin temp., venous hypertension & ischemia distally, cont. machinery murmur ^{bruit}.

* investigation: MRI angio, Duplex & Doppler.

- angiography done in case of decision to do embolization

* Treatment: - Excision & vascular repair done in large fistula
- for small one either follow up (if complicated → surg)
- Some advice embolization of fistula.

* Complication: cosmetic, Hge, thrombosis, distal ischemia, venous hypertension, high output cardiac failure

Branham's sign: it indicates slowing of heart rate as soon as fistula is compressed.